2 (Reiterated) A method according to claim 1 wherein said treatment of diabetes mellitus ameliorates hyperglycemia.

3. (Reiterated) A method according to claim 2 wherein gluconeogenesis is modulated.

4. (Reiterated) A method according to claim 3 wherein transcription of PEPCK is inhibited.

E1 5. (Twice Amended) A method according to claim 2 wherein transcription of the glucagon gene is inhibited.

6. (Reiterated) A method according to claim 1 wherein said individual is a human.

E2 7. (Amended) A method according to claim 1 wherein said contacting is accomplished by oral, intravenous, subcutaneous, intramuscular or intracutaneous mode of administration.

12. (Reiterated) A method for treating diabetes mellitus, comprising contacting an individual with an effective amount of a compound which disrupts complex comprising cyclic AMP response element binding protein (CREB) and CREB binding protein (CBP), said compound identified by a method comprising:

(a) contacting a modified host cell with a test compound, wherein said modified host cell comprises:

a first fusion protein comprising a GAL4 DNA binding domain, operatively associated with the kinase-inducible domain (KID) of CREB,

a second fusion protein comprising an activation domain, operatively associated with the CREB binding domain (KIX) of CBP, and

a reporter construct comprising a GAL4 response element operatively linked to a reporter gene; and

(b) selecting those test compounds which cause reduced expression of the reporter gene product, wherein said compounds are identified as disrupting complex comprising CREB and CBP.

17. (Reiterated) A method for treating diabetes mellitus, comprising contacting an individual with an effective amount of a compound which disrupts complex comprising cyclic AMP response element binding protein (CREB) and CREB binding protein (CBP), said compound identified by a method comprising:

(a) contacting a modified host cell with a test compound, wherein said modified host cell comprises:

a first fusion protein comprising an activation domain, operatively associated with the kinase-inducible domain (KID) of CREB,

a second fusion protein comprising a GAL4 DNA binding domain operatively associated with the CREB binding domain (KIX) of CBP, and

a reporter construct comprising a GAL4 response element operatively linked to a reporter gene; and

(b) selecting those test compounds which cause reduced expression of the reporter gene product, wherein said compounds are identified as disrupting complex comprising CREB and CBP.

18. (New) A method for modulating glucose metabolism in an individual, said method comprising contacting the individual with an effective amount of a compound which inhibits binding of CREB to CBP.

19. (New) A method according to claim 18 wherein said modulating glucose metabolism results in decreased serum glucose.